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Physiologic Hypertrophy: Effects on Left Ventricular Systolic Mechanics in Athletes

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Physiologic hypertrophy resulting from intense athletic participation has been reported to result in normal, reduced and augmented overall left ventricular performance. Rather than representing true differences in left ventricular contractility, these data may reflect the variable degree of ventricular dilation and increased wall thickness that occur with different types of exercise. As such, the resultant altered loading conditions may diminish the ability of the usual indexes of left ventricular function to accurately assess the left ventricular contractile state. Therefore, three groups of elite athletes with distinct patterns of myocardial hypertrophy were investigated utilizing recently developed load-independent contractility indexes. Age-matched control subjects (n = 33) were compared with 11 swimmers, 11 long-distance runners and 11 power lifters. Rest echocardiogram, phonocardiogram and calibrated carotid pulse tracing were used to calculate left ventricular dimensions, wall thickness, mass, fractional shortening, velocity of shortening and mean, peak and end-systolic wall stresses and the stress-time and minute stress-time integrals.

Compared with control subjects, all athletes had increased left ventricular mass, even when values were normalized for body surface area. Runners had a dilated left ventricle and normal wall thickness, swimmers had a mildly dilated ventricle with increased wall thickness and power lifters had normal cavity size with markedly increased wall thickness. Peak systolic wall stress was normal in runners and swimmers and reduced in power lifters, whereas end-systolic stress was low in swimmers and power lifters and normal in runners. The minute stress-time integral, a measure of myocardial oxygen consumption, was normal in runners and swimmers but was significantly reduced in lifters. In runners, fractional shortening was significantly reduced with normal velocity of shortening, whereas swimmers and power lifters had significant augmentation of fractional shortening and velocity of shortening. Examination of the rate-corrected velocity of shortening–end-systolic stress relation revealed normal contractility with augmented systolic performance due to reduced afterload in swimmers and power lifters. Comparison of runners and control subjects revealed normal afterload but reduced preload in runners, which was manifested as reduced fractional shortening with normal afterload and contractile state.

Physiologic hypertrophy results in marked alterations in left ventricular loading conditions with secondary changes in systolic performance. When load-independent indexes are employed, the left ventricular contractile state is found to be normal in young athletes despite markedly increased left ventricular mass. Different types of exercise are associated with distinct patterns of left ventricular hypertrophy and dilation, necessitating individual assessment of preload and afterload in the interpretation of indexes of left ventricular function.

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Intense athletic participation results in myocardial hypertrophy that can be disproportionate to the increase in body surface area. Similar degrees of left ventricular hypertrophy may be present in patients with aortic stenosis, coarctation of the aorta or systemic hypertension (1,2). These pathologic cases have demonstrated complex changes in preload, afterload and contractility that lead to the failure of the load-dependent ejection phase indexes (for example, ejection fraction, percent fractional shortening and velocity of fiber shortening) to accurately reflect intrinsic contractile state (2). In highly trained athletes the left ventricle also undergoes geometric and hemodynamic changes that may influence ventricular loading conditions in a manner that could

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diminish the reliability of traditional measures of left ventricular performance as indexes of myocardial contractility (3–12). This may explain the reports of normal, decreased and increased ventricular function in subjects with physiologic hypertrophy. Accordingly, we studied left ventricular performance in young elite swimmers, runners and power lifters using noninvasive load-independent indexes of left ventricular contractility. These indexes, which are based on physiologic events at end-systole, were used to elucidate the effects of distinct patterns of left ventricular hypertrophy on myocardial mechanics.

Methods

Study subjects. The study group comprised 33 athletes and 33 age-matched normal control subjects (age 16 to 29 years). There were 11 swimmers, 11 long-distance runners and 11 power lifters in the athlete group. Swimmers were recruited from a swim team with a training program of 25 to 30 h/week; all had more than 5 years' experience in competitive participation. Runners were members of a college track team participating in long-distance events; all ran more than 60 miles/week (average 90). Power lifters had all been participants in national competition within the prior 6 months and had more than 4 years of serious lifting experience. Control subjects were healthy, nonsedentary individuals who did not engage in a regular training program. All participants were free of known cardiovascular disease, were taking no cardioactive medications and had a normal physical examination.

Data recording. Data were collected using previously described methods (13–17). Echocardiograms were obtained using either a Hewlett-Packard 77020A two-dimensional ultrasound system with two-dimensionally directed M-mode capabilities or an Irex System II ultrasound module. High speed (100 mm/s) hard copy M-mode echocardiograms were obtained of the left ventricular minor axis with simultaneous phonocardiogram, electrocardiogram and indirect carotid pulse tracing. The phonocardiogram was recorded from the right upper sternal border. A Dinamap 845 or 1846P vital signs monitor (Critikon, Inc.) was used to obtain peak systolic and diastolic blood pressure measurements. Long- and short-axis views of the left ventricle were obtained with two-dimensional echocardiography for evaluation of regional wall motion in all participants except swimmers.

Data analysis. High quality tracings from each subject were selected for computer analysis on a Franklin Quantic 1200 echocardiographic review station (Bruce Franklin, Inc.). This device has a digitizing pad with a sampling rate of 80/cm, giving a net digitizing rate of 800 points/s. The carotid pulse tracing and the left ventricular echocardiogram, including the endocardial and epicardial borders of the posterior wall, were digitized. The carotid pulse tracing was corrected for time delay by aligning the dicrotic notch with the first high frequency component of the aortic component of the second heart sound.

From the digitized data, the following instantaneous measurements were derived by averaging three to five cardiac cycles: 1) left ventricular pressure throughout ejection, determined by linear interpolation using a calibrated carotid pulse tracing as previously described (13–17) (this method has been validated against an intraarterial standard in our laboratory [18]); 2) left ventricular internal diameter; 3) left ventricular posterior wall thickness; and 4) the left ventricular wall stress calculated from the angiographically validated formula (19):

$$WS = \frac{(P)(D)\ 1.35}{(h)\ [1 + (h/D)]\ (4)},$$

where WS is wall stress (g/cm^2), P is pressure (mm Hg), D is dimension, h is posterior wall thickness (cm) and 1.35 is the conversion factor from mm Hg to g/cm^2. Mean ejection wall stress was calculated from instantaneous wall stress values averaged over the period from the onset of ejection to aortic valve closure. The integral of the instantaneous stress-time relation was calculated for the ejection period to obtain the left ventricular stress-time integral, and the latter was multiplied by heart rate to obtain the left ventricular stress-time/min (17).

End-diastolic dimension and wall thickness were measured at the Q wave of the electrocardiogram, and end-systolic measurements were taken at the time of the first high frequency component of the second heart sound. The left ventricular percent fractional shortening was calculated as the difference between dimensions at end-diastole and end-systole, divided by the end-diastolic dimension (13). Left ventricular ejection time was measured from the simultaneous carotid pulse tracing and rate-corrected to a heart rate of 60 beats/min by dividing by the square root of the RR interval. The rate-corrected mean velocity of shortening was calculated by dividing fractional shortening by the rate-corrected ejection time (14).

Left ventricular mass was calculated using the modified formula of Devereux and Reichek (20):

$$\text{Mass} = 1.04\ [(D + 2h)^3 - D^3] - 14g,$$

where D and h represent end-diastolic dimension and wall thickness, respectively. Because left ventricular mass is directly proportional to body surface area and left ventricular dimension is linearly related to the cubic root of body surface area (21), left ventricular mass index and end-diastolic dimension index were calculated by dividing the nonindexed variables by body surface area and by the cube root of body surface area, respectively.

The relation of fractional shortening and rate-corrected velocity of shortening to end-systolic wall stress was determined for each individual and the mean values were obtained

for each of the four groups. These were then compared with the previously reported normal values for these indexes (13,14).

Statistical analysis. Data are reported as mean ± SD unless otherwise noted. Comparisons among the four groups were performed with one-way analysis of variance using the Tukey method for multiple comparison testing (22). A probability (p) value of <0.05 was considered statistically significant.

Results

Ventricular dimensions. Hemodynamic and echocardiographic data for the control and athlete groups are summarized in Table 1. Body surface area was greater in power lifters than it was in the other groups, and was moderately increased in swimmers compared with runners or control subjects, whereas runners and control subjects were not different from each other. Heart rate was significantly lower in runners. In all three groups of athletes, left ventricular mass was greater than that of control subjects, even when values were indexed for body surface area. Left ventricular mass in runners and swimmers did not differ significantly, whereas power lifters had a greater left ventricular mass and mass index compared with runners or swimmers. End-diastolic dimension was higher in all athletes than in control subjects, but after adjusting for body surface area, only runners and swimmers had a significantly increased end-diastolic dimension index. In contrast, the end-diastolic dimension index in power lifters was not different from that in the control group. The end-systolic dimension was also greater than control values in runners and swimmers. End-diastolic wall thickness was greater than the control value in all three groups of athletes, with power lifters having significantly greater values than those of runners or swimmers. Swimmers had greater end-systolic wall thickness than did runners or control subjects, and power lifters had higher values than those of each of the other three groups.

Blood pressure, wall stress, and ventricular function. Lifters had higher peak systolic, end-systolic and diastolic blood pressures than did any of the other groups. Peak systolic pressure was greater in swimmers than in runners or control subjects. End-systolic pressure was lower in runners than in swimmers or control subjects. Peak systolic

Table 1. Hemodynamic and Echocardiographic Data in the Four Study Groups

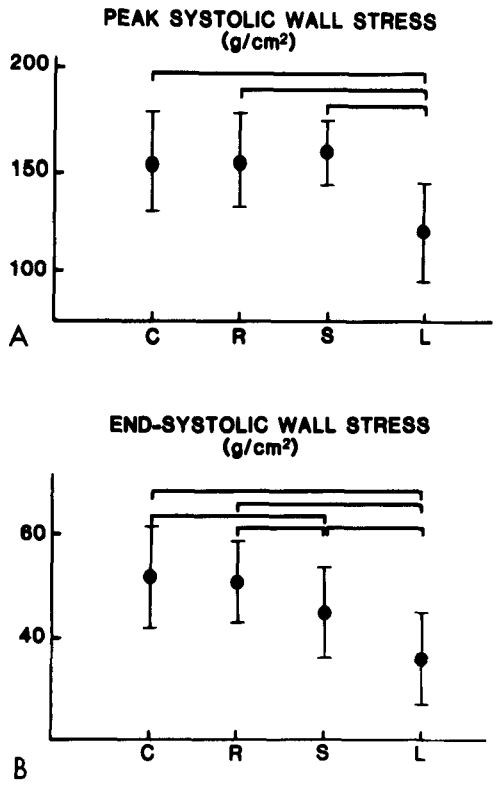
	Control Subjects	Runners	Swimmers	Lifters
Number	33	11	11	11
Age (yr)	22 ± 5.8	21 ± 2.3	21 ± 3.7	24 ± 2.8
BSA (m^2)	1.71 ± 0.22	1.70 ± 0.12	1.84 ± 0.20*†	2.28 ± 0.20*†‡
HR (beats/min)	72 ± 22	57 ± 7*	66 ± 13	74 ± 14†
EDD (cm)	4.8 ± 0.4	5.2 ± 0.3*	5.4 ± 0.5*	5.4 ± 0.7*
EDDI $cm^2/(m^2)^{1/3}$	4.0 ± 0.4	4.4 ± 0.3*	4.4 ± 0.4*	4.1 ± 0.6
ESD (cm)	3.2 ± 0.4	3.7 ± 0.3*	3.6 ± 0.4*	3.4 ± 0.4
FS (%)	32.2 ± 3.8	29.7 ± 2.9*	34.3 ± 2.6*†	37.4 ± 3.3*†‡
EDh (cm)	0.88 ± 0.13	0.99 ± 0.10*	1.00 ± 0.12*	1.32 ± 0.22*†‡
ESh (cm)	1.47 ± 0.20	1.40 ± 0.11	1.76 ± 0.13*†	2.19 ± 0.33*†‡
LVM (g)	168 ± 55	230 ± 50*	251 ± 73*	373 ± 125*†‡
LVMI (g/m^2)	98 ± 26	135 ± 25*	136 ± 35*	165 ± 57*†‡
PSP (mm Hg)	115 ± 10	114 ± 38	133 ± 10*†	151 ± 10*†‡
ESP (mm Hg)	98 ± 13	78 ± 6*	102 ± 17†	112 ± 12*†‡
DP (mm Hg)	66 ± 9	65 ± 4	66 ± 7	80 ± 9*†‡
Vcfc (circ/s)	1.01 ± 0.15	0.99 ± 0.11	1.08 ± 0.12*†	1.15 ± 0.13*†‡
ETc (ms)	318 ± 20	300 ± 12	318 ± 13	324 ± 12
ET (ms)	291 ± 18	308 ± 10	303 ± 11	293 ± 10
PSS (g/cm^2)	155 ± 24	156 ± 22	160 ± 15	121 ± 26*†‡
ESS (g/cm^2)	52 ± 10	51 ± 8	45 ± 9*†	36 ± 9*†‡
MS (g/cm^2)	95 ± 14	96 ± 8	98 ± 8	79 ± 14*†‡
ST (g/s per cm^2)	27 ± 5	29 ± 4	28 ± 5	21 ± 6*†‡
MST (g/s per cm^2 per min)	1,970 ± 550	1,650 ± 310	1,930 ± 660	1,500 ± 350*†‡

*p = 0.05 versus control; †p = 0.05 versus runners; ‡p = 0.05 versus swimmers. BSA = body surface area; CIRC = circumferences; DP = diastolic pressure; EDD = end-diastolic dimension; EDDI = end-diastolic dimension index; EDh = end-diastolic wall thickness; ESD = end-systolic dimension; ESh = end-systolic wall thickness; ESP = end-systolic pressure; ESS = end-systolic stress; ET = ejection time; ETc = rate-corrected ejection time; FS = fractional shortening; HR = heart rate; LVM = left ventricular mass; LVMI = left ventricular mass index; MS = mean stress; MST = minute stress-time; PSP = peak systolic pressure; PSS = peak systolic stress; ST = stress-time integral; Vcfc = rate-corrected velocity of shortening.

wall stress was significantly lower in power lifters than in the other three groups, and did not differ significantly among swimmers, runners and control subjects (Fig. 1A). End-systolic wall stress was significantly lower in swimmers and power lifters than in runners or control subjects, with power lifters having lower values than swimmers (Fig. 1B). Fractional shortening was lower in runners than in the other three groups, was elevated in swimmers compared with control subjects and was significantly higher in lifters compared with all three groups. Rate-corrected velocity of shortening was higher in power lifters than in swimmers, runners or control subjects and was higher in swimmers than in runners or control subjects.

Wall stress determinants. The determinants of end-systolic stress are displayed in Figure 2. Compared with control subjects, runners had a larger end-systolic dimension, lower end-systolic pressure and no difference in wall thickness, so that end-systolic stress was not different. In contrast, although swimmers had equivalent end-systolic pressure and larger end-systolic dimension than did control subjects, wall thickness was increased sufficiently to reduce end-systolic stress compared with the control value. Finally, power lifters had a higher end-systolic pressure, at similar end-systolic dimension, and a marked increase in end-systolic wall thickness resulting in a 31% decrease in end-systolic stress compared with the control value. Mean systolic left ventricular wall stress, ejection stress-time integral and stress-time/min relation were each significantly lower in the lifters than in control subjects. The swimmers and runners had mean stress, stress-time and minute stress-time values that were not different from those of control subjects.

Figure 1. Comparison of peak (**A**) and end-systolic (**B**) wall stress in control subjects (C), runners (R), swimmers (S) and power lifters (L). The superposed brackets indicate groups between which a significant ($p < 0.05$) difference was found. Lifters had reduced peak wall stress compared with control subjects, whereas swimmers and runners had values in an equivalent range. Significant reduction in end-systolic stress was found in both power lifters and swimmers but not in runners. Lifters had values for peak and end-systolic wall stress that were significantly lower than those for all other groups.



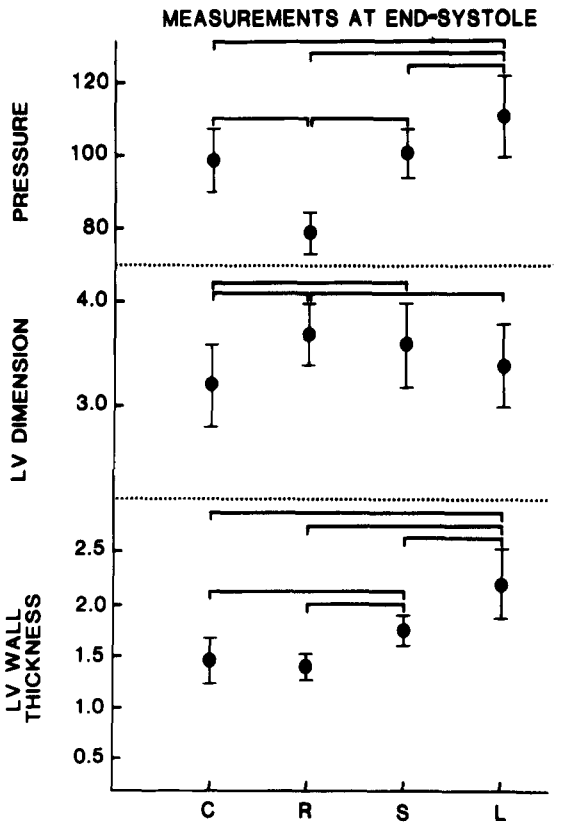


Figure 2. Comparison of the determinants of left ventricular (LV) end-systolic wall stress in control subjects (C), runners (R), swimmers (S) and power lifters (L). The superposed brackets indicate groups between which a significant ($p < 0.05$) difference was found. Reduced pressure was balanced by increased dimension with no change in wall thickness in runners, resulting in a normal end-systolic stress. Although dimension was increased in swimmers, thickness was disproportionately increased with normal pressure, resulting in reduced end-systolic stress. In lifters, greatly increased wall thickness more than compensated for the increased pressure, and dimension was not different from the control value, resulting in a significant reduction in end-systolic stress.

Role of afterload and preload. The relation of overall left ventricular systolic function to afterload (measured as end-systolic wall stress) is shown in Figure 3. For purposes of comparison, confidence intervals derived over a wide range of afterload conditions in normal subjects (13,14) are

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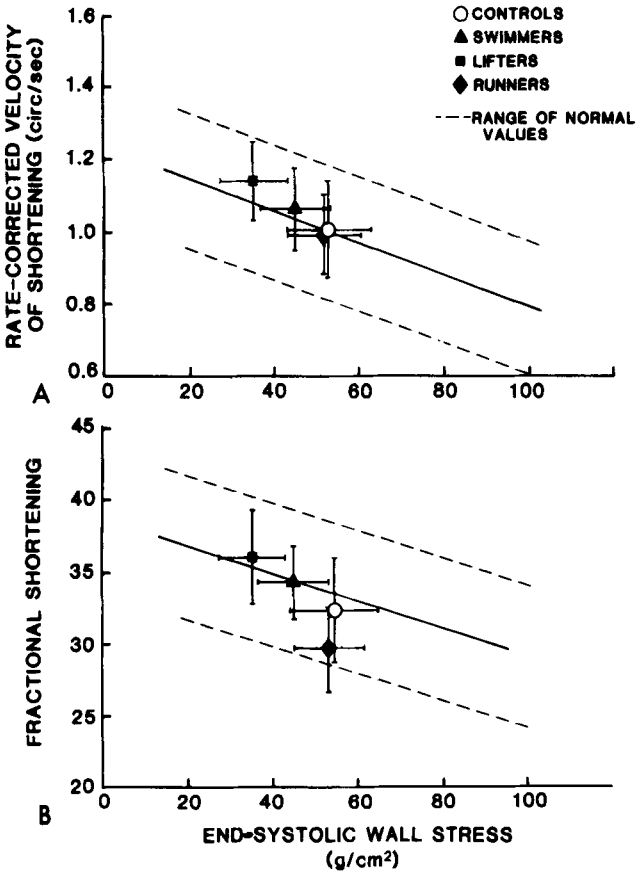

Figure 3. Relation of rate-corrected velocity of shortening (**A**) and fractional shortening (**B**) to left ventricular afterload as measured by end-systolic wall stress. The mean regression line and 95% confidence intervals for normal subjects in our laboratory are given for comparison. The increase in velocity of shortening and fractional shortening seen in swimmers and lifters was due to the reduced afterload in these subjects. Runners, in contrast, had normal values for the end-systolic stress–rate-corrected velocity of shortening relation with reduced values for the end-systolic stress-fractional shortening relation. This pattern has been previously shown to reflect reduced preload status (14).

included. Whereas both the end-systolic stress-fractional shortening and end-systolic stress–rate-corrected velocity of shortening relations incorporate afterload and are sensitive to left ventricular contractility, only the end-systolic stress–rate-corrected velocity of shortening relation is independent of preload (14). Thus, a normal end-systolic stress–rate-corrected velocity of shortening relation with an abnormal end-systolic stress–fractional shortening relation occurs only in the presence of altered preload status. The increased fractional shortening found in swimmers and power lifters was secondary to a proportional reduction in end-systolic stress (Fig. 3A). In contrast, runners demonstrated a reduction in fractional shortening in the absence of altered afterload (that is, end-systolic stress). When rate-corrected velocity of shortening is examined relative to the afterload conditions (Fig. 3B), all four groups fall within the normal range. Thus, the end-systolic stress–fractional shortening and the end-systolic stress–rate-corrected velocity of shortening data are concordant for normal subjects, swimmers and lifters, indicating normal preload status in all three groups and demonstrating that the increased systolic performance found in swimmers and power lifters is due to reduced afterload rather than augmented left ventricular contractility. However, the significant reduction in the end-systolic stress–fractional shortening relation in the runners is due to a relative reduction in preload because the preload-insensitive end-systolic stress–rate-corrected velocity of shortening relation is well within the predicted range for normal left ventricular contractile state (14).

Discussion

Aerobic versus anaerobic exercise. The patterns of hypertrophy seen in this study conform to previously recognized trends (2–12). Subjects who participate in predominantly aerobic exercise, such as long distance running, manifest an increase in left ventricular dimensions with relatively little change in wall thickness to dimension ratio. This parallels the findings in patients with volume-overload lesions of the left ventricle, and the cause is believed to be similar. That is, with prolonged exercise the left ventricle is faced with sustained elevation of preload due to increased venous return, leading to the addition of sarcomeres in series (23). In support of this hypothesis, a 28% increase in left ventricular mass associated with only a 7% increase in myocardial cell cross-sectional area has been observed in volume-overload ventricles (24). The net result is ventricular dilation with an increase in wall thickness that is proportional to the increase in dimension. In contrast, athletes engaged in short bouts of intense anaerobic exercise, such as power lifting, do not experience sustained elevation of cardiac output and therefore do not develop ventricular dilation. Left ventricular wall thickness increases in a fashion similar to that seen in pressure-overload cardiac disease (for example, systemic hypertension, aortic stenosis) with a relative increase in thickness to dimension ratio. This presumably is due to the higher exercise blood pressures associated with these activities. Indeed, intraarterial pressures as high as 480/350 mm Hg are found in power lifters during peak exercise (25). Swimmers were found to have an intermediate pattern with both left ventricular dilation and increased wall thickness. In this form of exercise, augmentation of cardiac output is less than it is in runners but greater than in power lifters, whereas pressor stress is greater than in runners and less than in power lifters (26–30). These athletes manifest cardiac structural changes that resemble those seen in combined pressure and volume overload.

Left ventricular wall stress: altered afterload and preload. To understand the observed differences in systolic performance among the four groups of subjects studied, it

is necessary to explore the concepts of preload and afterload. Although frequently approximated as arterial pressure or systemic vascular resistance, myocardial afterload is defined as the force per unit cross-sectional area in the left ventricular wall during contraction (19) and is best measured as wall stress.

Although it remains controversial which quantification of wall stress is the best measure of left ventricular afterload, recent work (2,17,19,23,31) suggests that peak systolic stress determines the extent of left ventricular hypertrophy whereas mean systolic stress and the integral of systolic stress with respect to time are most important as determinants of myocardial oxygen requirements. In contrast, it appears that end-systolic wall stress represents the limiting factor in myocardial fiber shortening (14) and is therefore the most relevant factor when issues concerning ventricular function are considered. Thus, power lifters and swimmers were observed to have low levels of end-systolic wall stress and therefore reduced afterload at rest. On the other hand, although preload is often approximated as end-diastolic volume or pressure, it is actually the degree of end-diastolic myofilament overlap that accounts for the Frank-Starling mechanism at the ultrastructural level (32). This will clearly be affected by the addition of new myofibers in series independent of any alteration in pressure or volume. Because direct measurement of filament overlap is not available, a functional measure that incorporates the effects of altered preload is needed. We have previously demonstrated (14) that in situations in which contractile state is constant, alteration in preload uniquely accounts for a divergent response of the degree of myocardial shortening compared with the velocity. This empiric difference permits assessments of preload status in a quantitative fashion in situations in which contractility is invariant.

Left ventricular systolic performance. Left ventricular systolic performance in our three groups of athletes was found to vary from depressed to supranormal, similar to the findings reported by others (1,5). The stress-velocity analysis performed here demonstrates that the variation in myocardial shortening characteristics is due not to altered contractile state, but rather to altered loading conditions caused by ventricular hypertrophy and dilation. Power lifters have normal cavity size with a marked increase in wall thickness and a secondary reduction of peak and end-systolic wall stress under rest conditions. Conceivably, these values would be normal under the pressor conditions of intense exercise such as power lifting. The elevation in fractional shortening and rate-corrected velocity of shortening that was found in these subjects is proportional to the reduction in afterload. The mechanism of augmented systolic performance is therefore the same as was previously reported (2) in young subjects with congenital valvular aortic stenosis. Contractile state, as measured by the end-systolic stress–rate-corrected velocity of shortening relation, is normal. Swimmers were found to have left ventricular functional characteristics intermediate between those of runners and power lifters, with dilation and a lesser degree of increased wall thickness than in lifters, resulting in less elevation in rate-corrected velocity of shortening and fractional shortening in proportion to the smaller reduction in afterload. Again the end-systolic stress–rate-corrected velocity of shortening relation indicated a normal contractile state. In contrast to both lifters and swimmers, runners have ventricular dilation without disproportionate increase in wall thickness, resulting in normal afterload. Therefore, rate-corrected velocity of shortening is normal in these subjects and the end-systolic stress–rate-corrected velocity of shortening relation again indicates a normal contractile state.

Mechanism of ventricular adaptation to exercise in athletes. The decrease in the end-systolic stress–fractional shortening relation with a normal end-systolic stress–rate-corrected velocity of shortening relation found in runners in this study is the expected finding in preload reduced states. This phenomenon reflects a left ventricle that is adapted to a high cardiac output during prolonged participation in aerobic exercise and is then assessed at rest (that is, during relatively reduced preload). The mechanism is parallel to that seen with preload reduction by means of venodilators or diuretics in patients with congestive cardiomyopathy, in whom a decrease in ejection fraction may be induced in spite of marked persistent left ventricular dilation. Further evidence for this mechanism is the observed greater percent increase in diastolic volume from rest values during exercise in endurance-trained athletes compared with control subjects (33), implying that at rest these athletes function on a lower portion of the passive diastolic pressure-volume curve. This finding also illustrates the limitations of utilizing a single measurement of end-diastolic volume as a measure of preload. At the sarcomere level, preload reflects the degree of filament overlap before the onset of contraction. Addition of sarcomeres in series permits a greater degree of filament overlap at the same end-diastolic volume. This appears to be the situation with these long-distance runners, in whom a reduced preload status is found at rest despite ventricular dilation. Finally, this observation supports the concept that heart rate reduction in athletes is the result of ventricular dilation rather than its cause. It has been suggested (34,35) that physical training leads to autonomic alterations that reduce rest heart rate, leading secondarily to ventricular enlargement. However, if bradycardia were the primary event, rest preload status would be expected to be either elevated or normal, in contrast to our observations of reduced rest preload status. Thus, it appears more likely that dilation is the primary event and reduction of heart rate is an adjustment to maintain a normal cardiac output in the presence of elevated stroke volume.

Myocardial oxygen consumption in athletes. Left ventricular minute stress-time was low at rest in athletes with

marked hypertrophy (lifters) and was normal in runners despite marked ventricular dilation. This index incorporates left ventricular systolic wall stress and heart rate, which represent two of the three major determinants of myocardial oxygen consumption. The remaining major determinant (contractile state) was found to be similar in the four groups of subjects. Thus, the compensatory hypertrophy in lifters results in low rest myocardial oxygen consumption (per gram of tissue) while protecting against excess demand during exercise. In contrast, runners manifest normal myocardial oxygen consumption at rest in spite of ventricular dilation, and function at a lower than normal point on the preload curve relating end-diastolic volume to stroke volume, permitting them to take greater advantage of the less energy-consuming Frank-Starling effect (33).

Prognostic role of physiologic ventricular hypertrophy. Although physiologic hypertrophy does not result in a depressed contractile state in young adults, other potential adverse effects of myocardial hypertrophy were not addressed in this study. Thus, hypertension-induced hypertrophy is associated with increased ectopic rhythm activity and an increased risk of sudden death when compared with findings in hypertensive patients without left ventricular hypertrophy (36–38). Reports of sudden death and an increased frequency of high grade ventricular arrhythmias in athletes have appeared (39–43). Decreased subendocardial capillary density has been noted in hypertrophy associated with both volume (25) and pressure overload lesions (44), suggesting the lack of an appropriate neovascular response to pathologic hypertrophy. Although contractility is normal in athletes, whether the marked hypertrophy associated with intense athletic participation results in an increased risk for arrhythmia requires further investigation.

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